Arterial Blood Gas Interpretation

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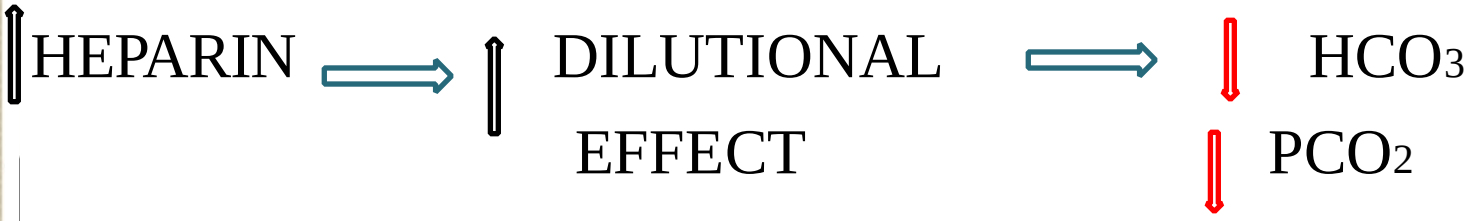
OBJECTIVES

- ABG Sampling
- Interpretation of ABG
  - Oxygenation status
  - Acid Base status
- Case Scenarios

ABG – Procedure and Precautions

- Ideally - Pre-heparinised ABG syringes
  - Syringe should be **FLUSHED** with 0.5ml of Heparin solution and emptied.

Do not leave excessive heparin in the Syringe



✓Only small 0.5ml Heparin for flushing and discard it
✓Syringes must have > 50% blood. Use only 2ml or less syringe

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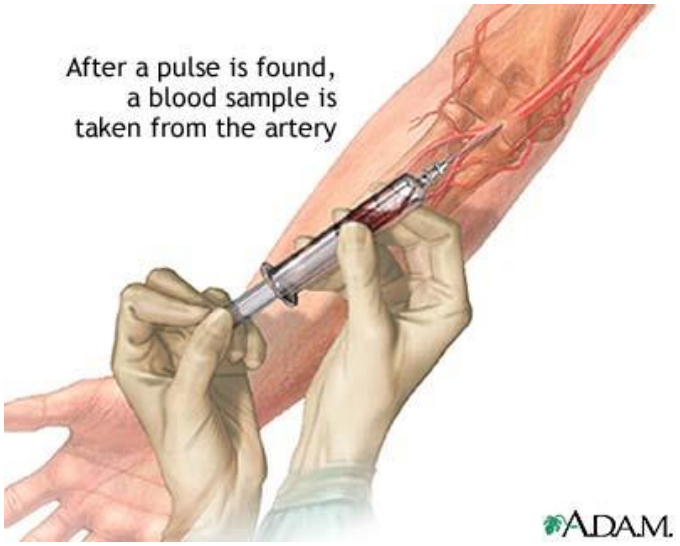
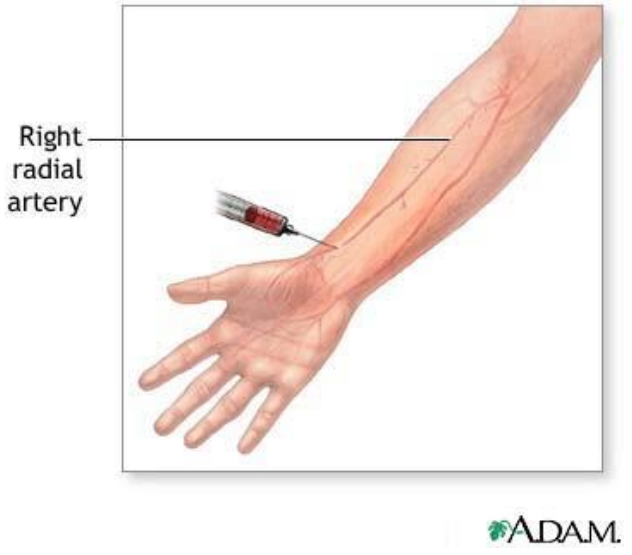
Sites for obtaining ABG

- Radial artery (most common)
- Brachial artery
- Femoral artery



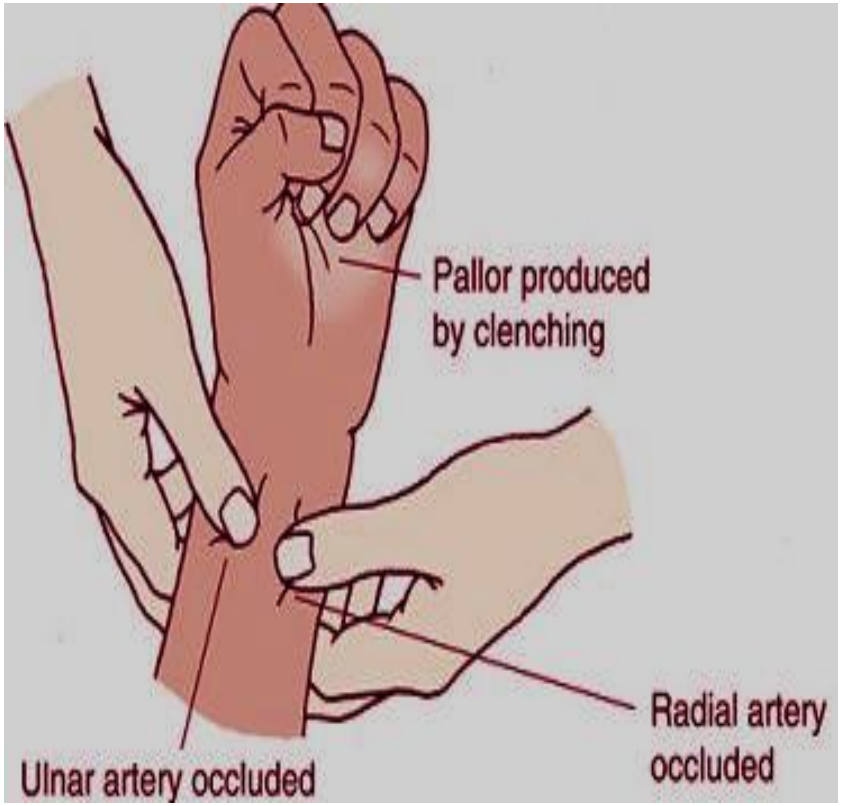
Radial is the <u>most preferable</u> site used because:

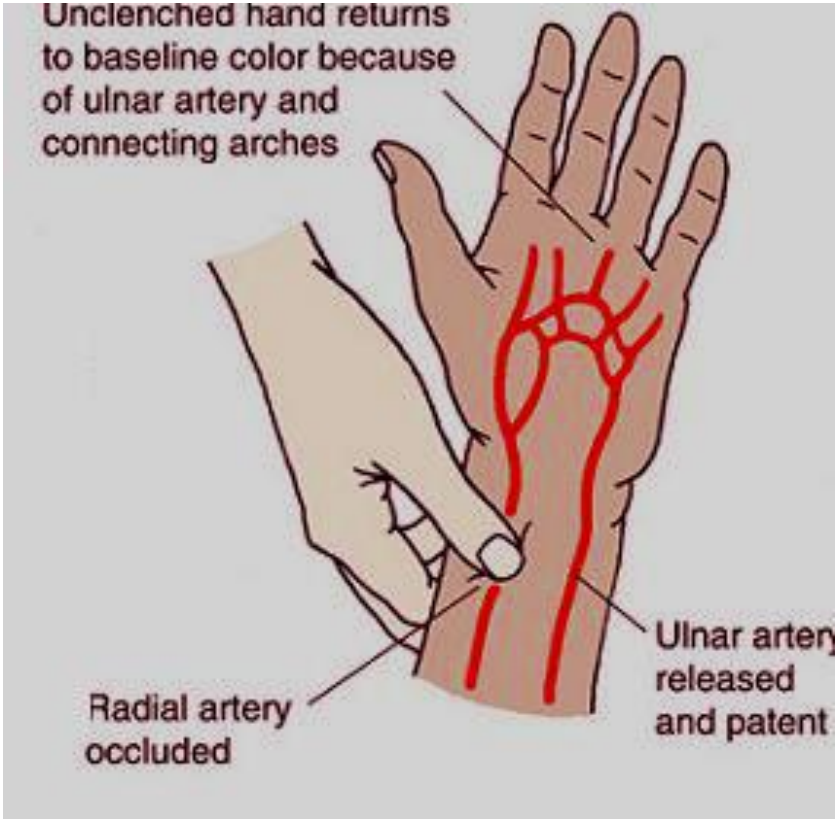
- It is easy to access
- It is not a deep artery which facilitate palpation, stabilization and puncturing
- The artery has a collateral blood circulation



ALLEN'S TEST

It is a test done to determine that collateral circulation is present from the ulnar artery





- Ensure No Air Bubbles. Syringe must be sealed immediately after withdrawing sample.
  - **Contact with AIR BUBBLES**

Air bubble = PO_2 150 mm Hg , PCO_2 0 mm Hg

Air Bubble + Blood = ↑ PO_2 ↓ PCO_2

- ABG Syringe must be transported at the earliest to the laboratory for **EARLY** analysis via **COLD CHAIN**

CHANGE IN VALUES EVERY 10 MINUTES	UNICED SAMPLE 37°C	ICED SAMPLE 4°C
pH	0.01	0.001
PCO_2	1 mm Hg	0.1 mm Hg
PO_2	0.1 %	0.01 %

Interpretation of ABG

- OXYGENATION
- ACID BASE

Blood Gas Report

- **Oxygenation Information**
- PaO_2 [oxygen tension]
- SaO_2 [oxygen saturation]

- **Acid-Base Information**
- PH
- $PaCO_2$
- HCO_3 [measured]

OXYGENATION

➢ Determination of PaO_2

PaO_2 is dependant upon ⟹ Age, FiO_2, P_{atm}

As **Age** ↑ the expected **PaO_2** ↓

- $PaO_2 = 109 - 0.4\,(Age)$

As **FiO_2** ↑ the expected **PaO_2** ↑

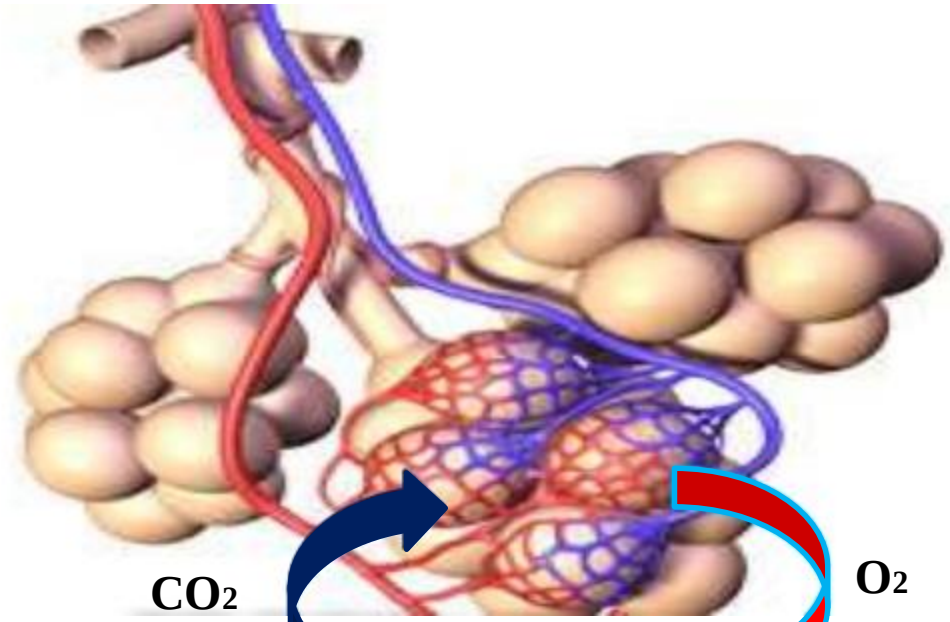
- Alveolar Gas Equation:
  - $P_AO_2 = (P_B - P_{H_2O}) \times FiO_2 - PaCO_2/R$

P_AO_2 = partial pressure of oxygen in Alveolar gas, **P_B** = Barometric Pressure (760mmHg), **P_{H_2O}** = water vapor pressure (47 mm Hg), **FiO_2** = fraction of inspired oxygen, **R** = respiratory quotient (0.8)

Determination of the PaO2 / FiO2 ratio

Inspired Air FiO_2 = 21%
PiO_2 = 150 mmHg

$P_{alv}O_2$ = 100 mmHg



PaO_2 = 95 mmHg

PaO_2/ FiO_2 ratio

- Gives understanding Patient Oxygenation with Respect to Oxygen delivered, more important than simply the PaO_2 value.

Example,

	Patient 1 On Room Air	**Patient 2 On MV**
PaO2	68	90
FiO2	21% (0.21)	50% (0.50)
P:F Ratio	324 ⬆	180 ⬇

HYPOXIA

VERSUS

HYPOXEMIA

HYPOXIA	HYPOXEMIA
Hypoxia is defined as a reduction of oxygen supply at the tissue level, which is not measured directly by a laboratory value	Hypoxemia is defined as a condition where arterial oxygen tension or partial pressure of oxygen (PaO_2) is measured to be between 80 and 100 mmHg
Patients may not indicate signs of hypoxemia	Patients will also experience hypoxia

CLASSIFICATION OF HYPOXEMIA

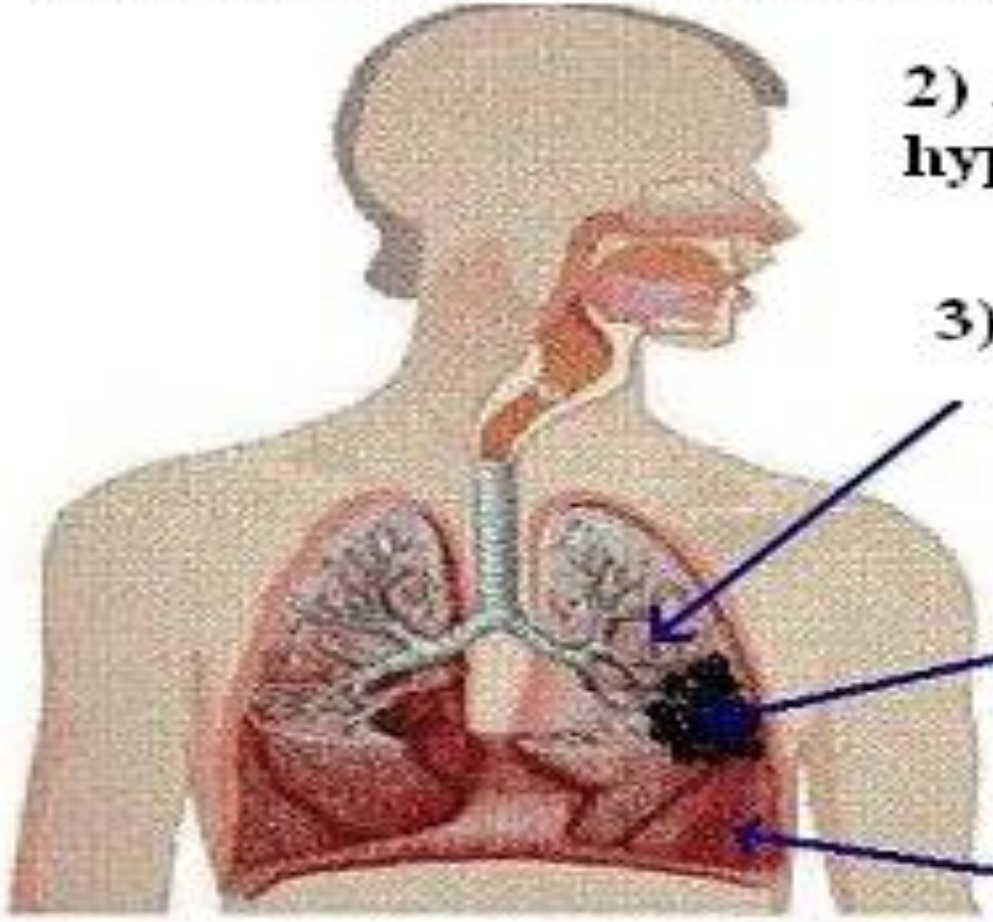
Classifications	PaO_2 (rule of thumb)
Normal	80-100 mm Hg
Mild hypoxemia	60-80 mm Hg
Moderate hypoxemia	40-60 mm Hg
Severe hypoxemia	<40 mm Hg

This classification is based on predicted ***normal values for a patient who is less than 60 years old and breathing room air.*** For older patients, ***subtract 1 mm Hg for every year over 60 years of age*** from the limits of mild and moderate hypoxemia.

A PaO2 of less than 40 mm Hg represents severe hypoxemia at any age.

Causes of hypoxemia

1) Reduced partical pressure of oxygen in the inspired air

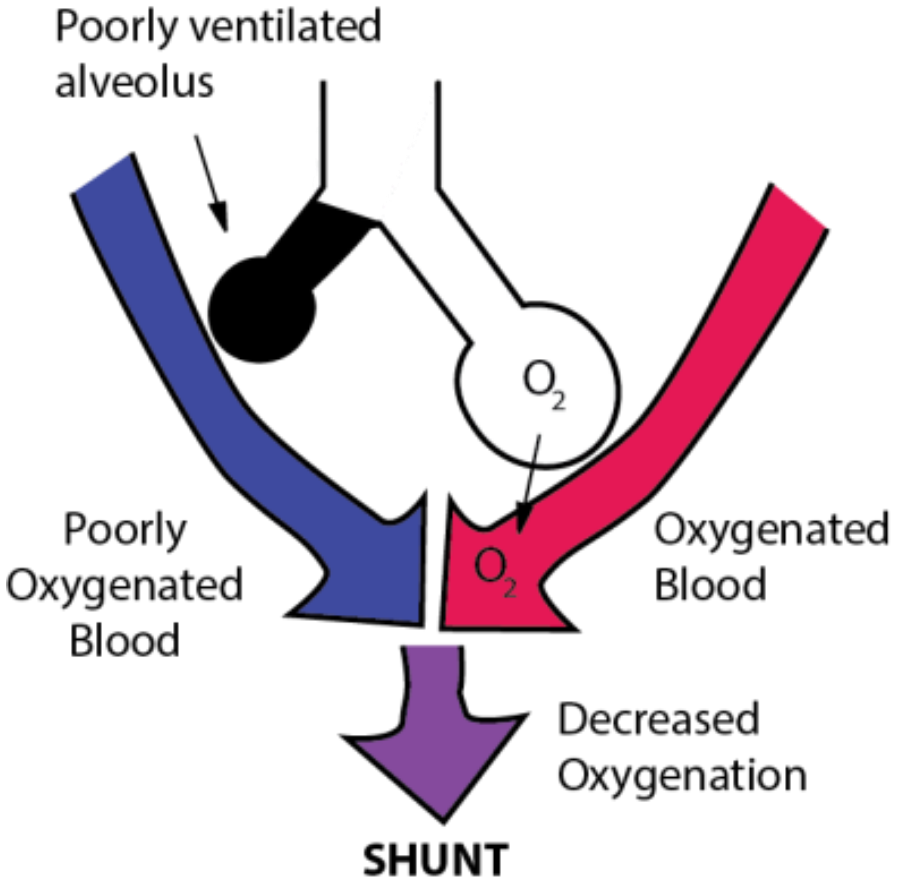


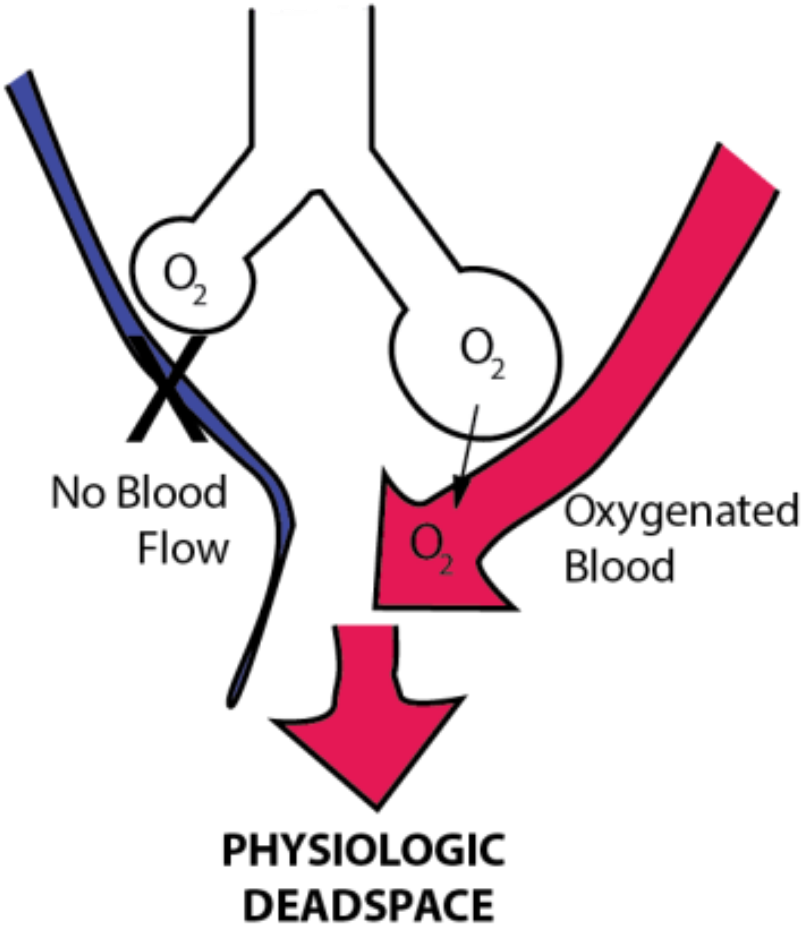
2) Alveolar hypoventilation

3) Ventilation-perfusion mismatch

4) Shunt (intracardiac or intrapulmonary)

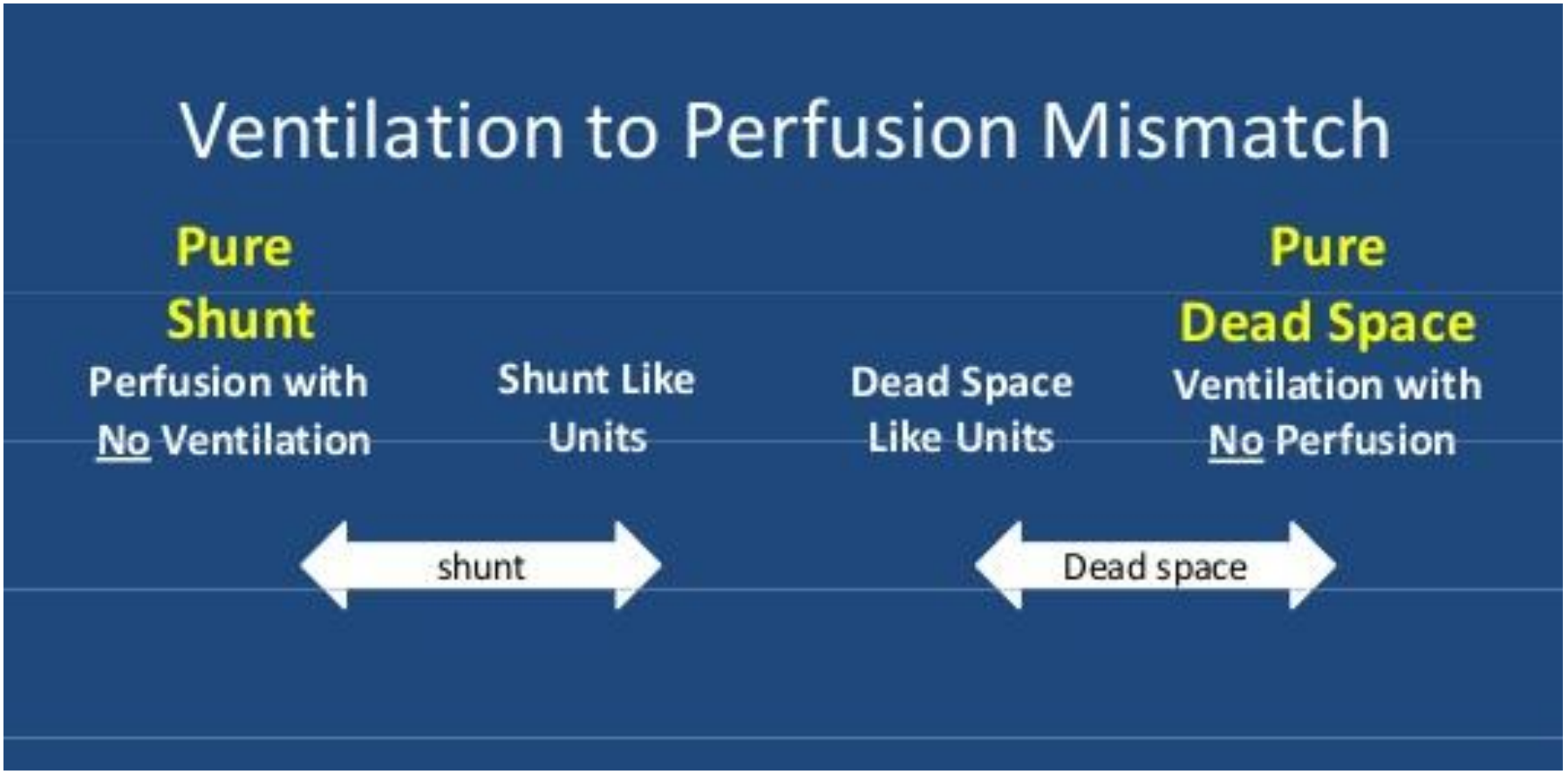
5) Impaired alveolar-capillary diffusion

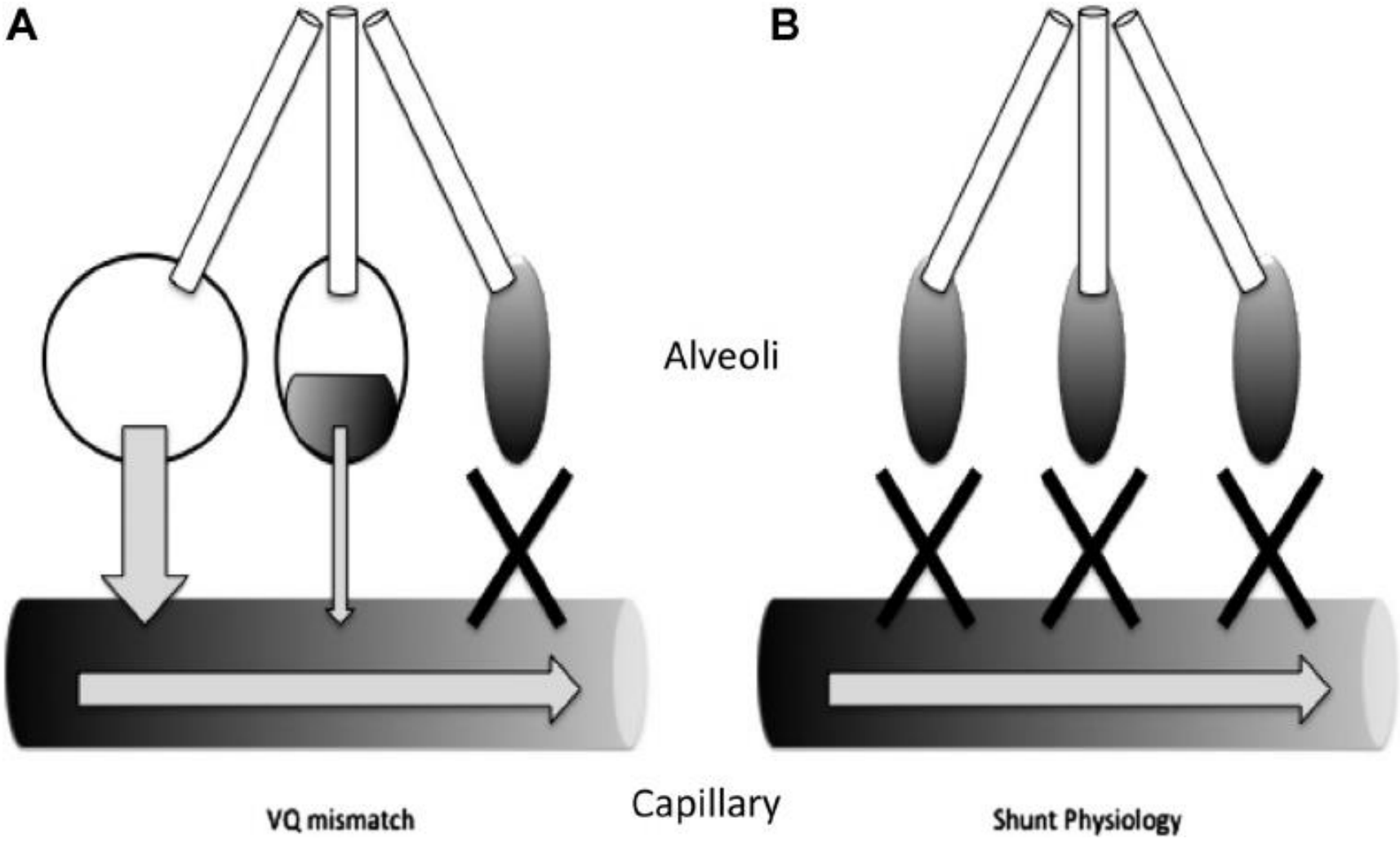




Shunt is perfusion of poorly ventilated alveoli.
Physiologic dead space is ventilation of poor perfused alveoli.

Ventilation to Perfusion Mismatch




A
B
Alveoli
Capillary
VQ mismatch
Shunt Physiology

The A-a gradient

A-a gradient = $P_AO2 - P_aO2$

Normal = < 15mmHg

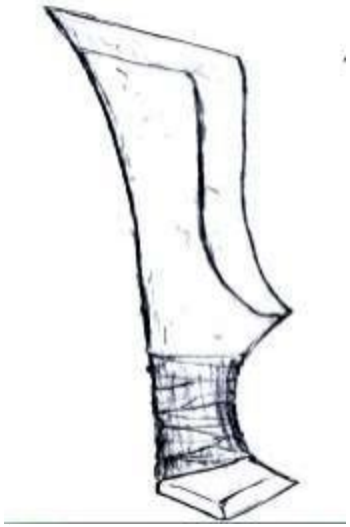
Normal rises 1mmHg per decade

CAUSE	P_{aO2}	A – a GRADIENT	EFFECT OF SUPPLEMENTAL O2
High Altitude	Decreased	Normal	Improves
Hypoventilation	Decreased	Normal	Improves
Diffusion Defect	Decreased	Increased	Improves
V°/Q Defect	Decreased	Increased	Improves
R → L Shunt	Decreased	Increased	Does not

A C I D B A S E

Acid Base Balance

- H^+ ion concentration in the body is precisely regulated
- The body understands the importance of H^+ and hence devised DEFENCES against any change in its concentration-



BICARBONATE BUFFER SYSTEM
Acts in few seconds

RESPIRATORY REGULATION
Acts in few minutes

RENAL REGULATION
Acts in hours to days

Assessment of ACID BASE Balance

- Definitions and Terminology

- **ACIDOSIS** – presence of a process which tends to ↓ pH by virtue of gain of H^+ or loss of HCO_3^-
- **ALKALOSIS** – presence of a process which tends to ↑ pH by virtue of loss of H^+ or gain of HCO_3^-

If these changes, change pH, suffix 'emia' is added

- **ACIDEMIA** – reduction in arterial pH (pH<7.35)
- **ALKALEMIA** – increase in arterial pH (pH>7.45)

Causes of Acid-Base Balance

Metabolic Acidosis Diabetic ketoacidosis Diarrhea Renal failure Shock Aspirin overdose Sepsis	**Metabolic Alkalosis** Loss of gastric secretions Overuse of antacids K+ wasting diuretics
Respiratory Acidosis Hypoventilation COPD Airway obstruction Drug overdose Chest trauma Pulmonary edema Neuromuscular disease	**Respiratory Alkalosis** Hyperventilation Hypoxia Anxiety High altitude Pregnancy Fever

Compensatory responses and their mechanisms.

Primary disorder	Primary Chemical change	Compensatory response	Compensatory Mechanism
Metabolic Acidosis	↓ HCO_3^-	↓ PCO_2	Hyperventilation
Metabolic Alkalosis	↑ HCO_3^-	↑PCO_2	Hypoventilation
Respiratory Acidosis	↑PCO_2	↑HCO_3^-	
Acute			Intracellular Buffering
Chronic			Renal Generation of HCO_3^-
Respiratory Alkalosis	↓ PCO_2	↓HCO_3^-	
Acute			Intracellular Buffering
Chronic			Renal secretion of HCO_3^-

If PCO_2 & $[HCO_3]$ move in opposite directions

Normal Values

ANALYTE	Normal Value	Units
pH	**7.35 - 7.45**	
PCO2	**35 - 45**	mm Hg
PO2	**80 – 100**	mm Hg`
[HCO3]	**22 – 26**	meq/L
SaO2	**95-100**	%
Anion Gap	**12±4**	meq/L
ΔHCO3	**+2 to -2**	meq/L

Step Wise Approach to Interpretation of ABG Reports

STEP 1

- Acidosis or Alkalosis?

STEP 2

- Respiratory or Metabolic?

STEP 3

- If Resp. Acute or Chronic and Compensation

STEP 4

- If Metabolic, Compensation and Anion gap?

Step 1 Acidosis, Alkalosis, or normal?

- PH is < 7.35, ⇛ Primary process is acidosis.
- PH is > 7.45, ⇛ Primary process is alkalosis.

Step 2: Is the primary disturbance Respiratory or Metabolic?

Look at the paCO2 and pH

If both go with the same direction the primary disturbance is ⇛ Metabolic

If both go with different direction the primary disturbance is ⇛ Respiratory

Step 3: For Primary Respiratory disturbance, is it acute or chronic? then Compansation

Acute or chronic

PaCO2 and pH

Acute condition.

for each 1mm Hg PaCO2 ⇛ pH changes 0.008 .

pH changes (Δ pH) = 0.008 × $\Delta PaCO_2$

Chronic condition.

for each 1mm Hg PaCO2 ⇛ pH changes 0.003

pH changes (Δ pH) = 0.003 × $\Delta PaCO_2$

IF RESPIRATORY, IS IT ACUTE OR CHRONIC?

- Acute respiratory disorder - $\Delta pH_{(e\text{-}acute)} = 0.008x\ \Delta Pco_2$
- Chronic respiratory disorder - $\Delta pH_{(e\text{-}chronic)} = 0.003x\ \Delta pCO_2$

- Compare, $pH_{measured}\ (pH_m)$ v/s $pH_{expected}\ (pH_e)$

$pH_{(m)} = pH_{(e\text{- acute})}$	$pH_{(m)}$ = between $pH_{(e\text{- acute})}$ & $pH_{(e\text{- chronic})}$	$pH_{(m)} = pH_{(e\text{-chronic})}$
ACUTE RESPIRATORY DISORDER	PARTIALLY COMPENSATED	CHRONIC RESPIRATORY DISORDER

Step 3: For Primary Respiratory disturbance, is it acute or chronic? then Compansation

Compansation

PaCO2 and HCO3

Respiratory acidosis:

- Acute condition.

  for each 10mm Hg PaCO2 ↑ ⇛ HCO3 ↑ by 1 meq.

- Chronic condition.

  for each 10mm Hg PaCO2 ↑⇛ HCO3 ↑by 4 meq

Respiratory alkalosis:

- Acute condition.

for each 10mm Hg PaCO2 ↓ ⇛ HCO3 ↓ by2 meq.

- Chronic condition.

for each 10mm Hg PaCO2 ↓ ⇛ HCO3 ↓ by5 meq.

	acidosis	alkalosis
acute	1	2
chronic	4	5

a. Respiratory acidosis

Phase	PH	PaCO2	HCO3
UNCOMPENSATED	↓	↑	------

Because there is no response from the kidneys yet to acidosis the HCO3 will remain normal

Phase	PH	PaCO2	HCO3
PARTIAL COMPENSATED	↓	↑	↑

The kidneys start to respond to the acidosis by increasing the amount of circulating HCO3

Phase	PH	PaCO2	HCO3
FULL COMPENSATED	N	↑	↑

PH return to normal PaCO2 & HCO3 levels are still high to correct acidosis

B. Respiratory alkalosis

Phase	PH	PaCO2	HCO3
UNCOMPENSATED	↑	↓	------

Because there is no response from the kidneys yet to acidosis the HCO3 will remain normal

Phase	PH	PaCO2	HCO3
PARTIAL COMPENSATED	↑	↓	↓

The kidneys start to respond to the alkalosis by decreasing the amount of circulating HCO3

Phase	PH	PaCO2	HCO3
FULL COMPENSATED	N	↓	↓

PH return to normal PaCO2 & HCO3 levels are still low to correct alkalosis

Step4: For a metabolic disturbance, is the respiratory system compensating OK?

Metabolic acidosis

Expected $PCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$

Winter's Equation

Metabolic alkalosis

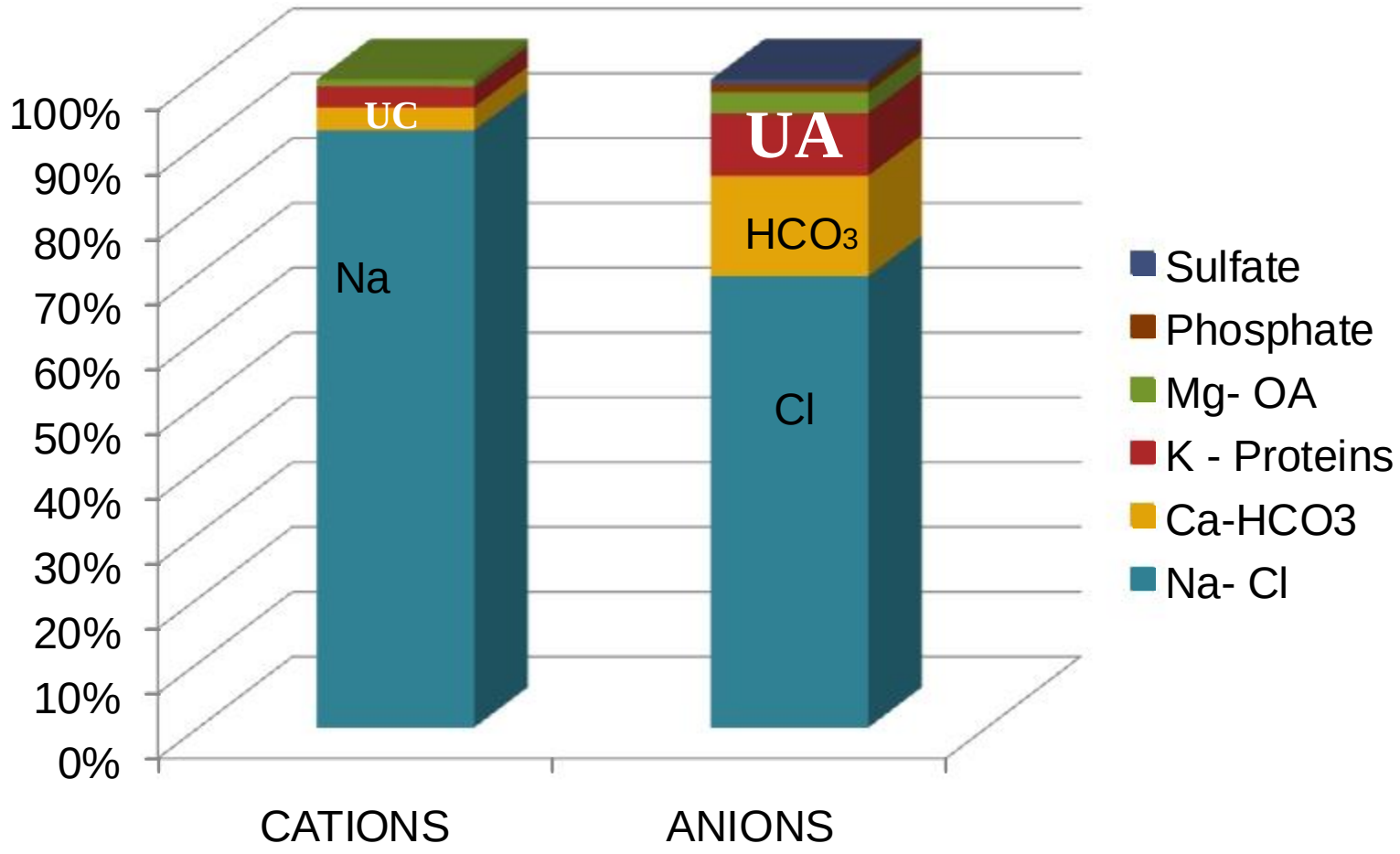
Expected $PCO_2 = 40 + (0.6 \times \Delta HCO_3^-)$

Quick rule of thumb : PCO_2 = last 2 digits of pH

For any metabolic disorder

Step4: For a metabolic acidosis, Anion gap?

Electrochemical Balance in Blood



METABOLIC ACIDOSIS-
ANION GAP?

IN METABOLIC ACIDOSIS WHAT IS THE ANION GAP?

- **ANION GAP(AG) = (Na + K) – (HCO_3 + Cl)**

Normal Value = 12 ± 4 (7- 16 Meq/l)

Metabolic Acidosis
- High Anion Gap Metabolic Acidosis
- Normal Anion Gap Acidosis

CAUSES OF METABOLIC ACIDOSIS
(High anion gap)→(Normochloremic)

- **LACTIC ACIDOSIS**
- **KETOACIDOSIS**
  - ✓ Diabetic
  - ✓ Alcoholic
  - ✓ Starvation
- **RENAL FAILURE**
  (acute and chronic)
- **TOXINS**
  - ✓ Ethylene glycol
  - ✓ Methanol
  - ✓ Salicylates
  - ✓ Propylene glycol

Normal anion gap(Hyperchloremic) MET.ACIDOSIS causes

❖ Gastrointestinal bicarbonate loss

A. Diarrhea

B. External pancreatic or small-bowel drainage

C. Ureterosigmoidostomy, jejunal loop, ileal loop

D. Drugs

1. Calcium chloride (acidifying agent)
2. Magnesium sulfate (diarrhea)
3. Cholestyramine (bile acid diarrhea)

❖ Renal acidosis

A. Hypokalemia

1. Proximal RTA (type 2)
2. Distal (classic) RTA (type 1)

B. Hyperkalemia

❖ Drug-induced hyperkalemia (with renal insufficiency)

A. Potassium-sparing diuretics (amiloride, triamterene, spironolactone)

B. Trimethoprim

C. Pentamidine

D. ACE-Is and ARBs

E. Nonsteroidal anti-inflammatory drugs

F. Cyclosporine and tacrolimus

❖ Other

A. Acid loads (ammonium chloride, hyperalimentation)

B. Loss of potential bicarbonate: ketosis with ketone excretion

C. Expansion acidosis (rapid saline administration)

Anion Gap and Albumin

- The normal AG is affected by patients plasma albumin concentration.

- For every 1g/dl reduction in plasma albumin concentration the AG decreases by 2.5

- **Corrected AG = Calculated AG + [2.5 × (4 – albumin)]**

- A patient with poorly controlled IDDM missed his insulin for 3 days.

pH 7.1 HCO_3 8 mEq/l $PaCO_2$ 20 mmhg Na 140 mEq/l CL 106 mEq/l and urinary ketones +++

Analysis

- pH is low so patient has **acidosis**. Low HCO3 is suggestive of **metabolic acidosis**. PaCO2 is also low suggestive of compensation.
- Expected compensation (fall in PaCO2) will be

PaCO2= HCO3 X 1.5 +8=8 X1.5 +8=12+8=20

- SO expected PaCO2 will be 20 mmhg, which matches with actual PaCO2, suggestive of simple ABD.
- AG is 26 (AG=Na-(Cl+HCO3)=140-(106+8)=140-114=26, which is high, S/o high AG Metabolic Acidosis. Presence of urinary ketones suggests presence of diabetic ketoacidosis.
- So the patient has **high anion gap metabolic acidosis** due to DKA

- ABG of patient with stable CHF on furosemide is as follows

pH 7.48 HCO_3 34 mEq/l $PaCO_2$ 48 mmhg

- **pH is high** so patient has **alkalosis.**
- **HCO3 is high** S/O **metabolic alkalosis.**
- PaCO2 is high, S/O compensation *(follows same direction rule)*
- Expected compensation (rise in PaCO2) will be

- Expected PCO2 = 40 + (0.6 X ΔHCO3-)

- ΔHCO3-= 34-24 = 10 mEq/L

So,Change in PaCo2 = 40 + (10x 0.6) = 46 mmHg, which almost matches with actual PaCO2 which is 48 mEq/L, Suggestive of simple ABD.

- So patient has **primary metabolic alkalosis due to diuretics.**

- Following sleeping pills ingestion, patient presented in drowsy state with sluggish respiration with respiratory rate 4/min.

pH 7.1 HCO_3 28 mEq/L $PaCO_2$ 80 mmhg PaO_2 42 mmhg

- **pH is low** so patient **has acidosis**.
- **High PaCO2** is S/O **respiratory acidosis**.
- Low PaO2 –hypoxia, supports diagnosis of respiratory failure- acidosis. HCO3 is also high suggestive of compensation *(same direction rule)*.

Is it Acute OR chronic respiratory disorder???

IN ACUTE (Δ pH) = 0.008 × Δ**PaCO2**= 0.008 × **(80-40)= 0.32**

IN CHRONIC (Δ pH) = 0.003× Δ**PaCO2**= 0.003 × **(80-40)= 0.12**

Δ pH = 7.4 – 7.1 = 0.3……. So It is Acute Disorder

PH ↓ CO2 ↑ HCO3 ↑ .....HCO3 increased for compensation but PH is still abnormal so there is partial compensation

- So, the patient has **Acute respiratory acidosis partially compensated due to respiratory failure,** due to sleeping pills.

Clinical correlation: Example 1

- A 15 year old boy is brought from examination hall in apprehensive state with complain of tightness of chest.

pH 7.54 HCO_3 21 mEq/L $PaCO_2$ 21 mm of hg

Example 1 : Analysis

- **pH is high** so patient has **alkalosis.**
- **Low PaCo2** is suggestive of **respiratory alkalosis.**

Is it Acute OR chronic respiratory disorder???

IN ACUTE (Δ pH) = 0.008 × Δ**PaCO2**= 0.008 × **(40-21)= 0.15**

IN CHRONIC (Δ pH) = 0.003× Δ**PaCO2**= 0.003 × **(40 -21)= 0.057**

Δ pH=7.54 - 7.4 = 0. 14..... **So It is Acute Disorder**

HCO3 decreased for compensation but PH is still abnormal so there is partial compensation

- So the patient has acute respiratory alkalosis partialy compensated due to anxiety.

- A case of hepatic failure has persistent vomiting

pH 7.54 **HCO3** 38 mEq/L **PaCO2** 44 mmhg

pH is high so patient has alkalosis. HCO3 is high S/O metabolic alkalosis (due to vomiting). PaCO2 is high suggestive of compensation (follows same direction rule)

- Expected compensation (rise in PaCO2) will be

Rise in PaCO2= 0.6 X rise in HCO3= 0.6 X (38-24) =0.6 X14=8.4

- So expected PaCO2 will be 40+8.4 =48.4 mmhg. But actual value of PaCO2 is lesser than expected PaCO2 (44 vs 48.4 mmhg) which suggests presence of additional respiratory alkalosis (hepatic failure can cause respiratory alkalosis).

- So, patient has mixed disorder, metabolic alkalosis with respiratory alkalosis.

CASE 1

62 years old Male patient

- COPD
- Breathlessness, progressively increased , aggravated on exertion, 2 days
- Chronic smoker
- expiratory rhonchi

22/7/2011	7:30 am
pH	7.20
PCO2	92 mmHg
PO2	76 mmHg
Actual HCO3	28.00 mmol/l
SO2	89
FiO2	37%

- STEP 1 – ACIDEMIA
- STEP 2 – pH ⇩ PCO_2 ⇧ Respiratory

- STEP 3 PH expected
  PH acute=7.40 - 0.008(92 - 40) =6.984
  PH chronic=7.40 - 0.003(92 - 40) =7.244
  PH (7.2) b/w 6.984 to 7.244

Primary Respiratory Acidosis,
partially compensated

STEP 4

Mild hypoxemia

CASE 2

63 years old ,Male patient

- CRF
- Breathlessness
- Decreased Urine Otpt. 2days
- Vomiting 10-15

31/7/2011	11:30pm
pH	7.18
PCO2	21.00
PO2	82
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
K	4
Albumin	2.4

31/7/11	11:30pm
pH	7.18
PCO2	21.00
PO2	90
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
K	4
Albumin	2.4

- STEP 1 – ACIDEMIA
- STEP 2 – pH ⬇ PCO2 ⬇
  METABOLIC

- STEP4 – PCO2 expected

$PCO2 = (1.5 \times HCO3) + 8 \pm 2$

$(1.5 \times 7.8) + 8 \pm 2 =$

$19.7 \pm 2 = 17.7 - 21.7$

- STEP5 ANION GAP

$= (Na + K) – (HCO3 + Cl)$

$= (140.6 + 4) - (7.80 + 102)$

$= 34.8$

HIGH AG Met. Acidosis


Thank you!
grammarly